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=> egfr
L1       35311 EGFR

=> "epidermal growth factor receptor"
L2       53835 "EPIDERMAL GROWTH FACTOR RECEPTOR"

=> l1 or l2
L3       62793 L1 OR L2

=> "cytotoxic t lymphocyte"
L4       48904 "CYTOTOXIC T LYMPHOCYTE"

=> l3 and l4
L5          79 L3 AND L4

=> l5 and 1970-2005/py
L6          55 L5 AND 1970-2005/PY

=> l6 and antibod?
L7          29 L6 AND ANTIBOD?

=> dup rem l7
PROCESSING COMPLETED FOR L7
L8          18 DUP REM L7 (11 DUPLICATES REMOVED)

=> l8 and peptide
L9          10 L8 AND PEPTIDE

=> kyogo?/au and itoh?/au
L10         19 KYOGO?/AU AND ITOH?/AU

=> shigeki?/au and shichijo?/au
L11          0 SHIGEKI?/AU AND SHICHIJO?/AU

=> l9 and l10
L12          0 L9 AND L10

=> d ti abs so l9 1-10

L9   ANSWER 1 OF 10     MEDLINE on STN
TI   Identification of   ***epidermal***     ***growth***     ***factor***
       ***receptor***  -derived   ***peptides***   recognised by both cellular
     and humoral immune responses in HLA-A24+ non-small cell lung cancer
     patients.
AB   The   ***epidermal***     ***growth***     ***factor***     ***receptor***
     (  ***EGFR***  ) is one of the most appropriate target molecules for
     cancer therapy because of its high expression in epithelial cancers.  A
     novel   ***EGFR***  -tyrosine-kinase inhibitor, ZD1839, has been approved
     as a drug for non-small cell lung cancer (NSCLC), and many other agents
     are now being tested in clinical trials.    ***Cytotoxic***     ***T***
       ***lymphocyte***   (CTL)-directed epitope   ***peptides***   could be
     another class of useful compounds in   ***EGFR***  -targeted therapies.
     However, at present, there are no data on CTL-directed   ***peptides***
     of   ***EGFR***  .  Therefore, this study aimed to identify immunogenic
       ***EGFR***  -derived   ***peptides***   in HLA-A24(+) NSCLC patients.  We
     report in this study three such   ***EGFR***  -derived   ***peptides***
     at positions 54-62, 124-132 and 800-809.  These   ***peptides***   were
     recognised by both cellular and humoral immune responses in most of the
     peripheral blood mononuclear cells (PBMCs) and sera from NSCLC patients
     that we tested.  These results may provide a scientific basis for the
     development of   ***EGFR***  -based immunotherapy.
SO   European journal of cancer (Oxford, England : 1990),   ***(2004 Jul)***
     Vol. 40, No. 11, pp. 1776-86.
     Journal code: 9005373. ISSN: 0959-8049.

L9   ANSWER 2 OF 10     MEDLINE on STN
TI   Identification of   ***epidermal***     ***growth***     ***factor***
       ***receptor***  -derived   ***peptides***   immunogenic for HLA-A2(+)
     cancer patients.
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AB ***Epidermal*** ***growth*** ***factor*** ***receptor*** (***EGFR***) is one of the most appropriate target molecules for cancer therapy because of its relatively high expression in about one-third of all epithelial cancers in correlation with neoplasmic progression. With respect to ***EGFR*** -targeted therapies, ***antibodies*** and tyrosine-kinase inhibitors have been intensively studied, a novel ***EGFR*** -tyrosine-kinase inhibitor ZD1839 has been approved as an anticancer drug, and many other agents are now under clinical trial. In addition, ***cytotoxic*** ***T*** ***lymphocyte*** (CTL)-directed epitope ***peptides*** could be another class of compounds useful in ***EGFR*** -targeted therapies. However, there is presently no information on CTL-directed ***peptides*** of ***EGFR*** . Therefore, from the viewpoint of development of ***peptide*** -based cancer therapy, this study was intended to determine the ***EGFR*** -derived ***peptides*** recognised by both cellular and humoral immunities in HLA-A2(+) epithelial cancer patients. We herein report finding of two such types of ***EGFR*** -derived ***peptides*** at position 479-488 and 1138-1147, both of which were recognised by the majority of patients' sera (IgG), and also possessed the ability to induce HLA-A2-restricted ***peptide*** -specific CTLs against ***EGFR*** -positive tumour cells in peripheral blood mononuclear cells (PBMCs) of epithelial cancer patients. These results may provide a scientific basis for the development of ***EGFR*** -based immunotherapy for HLA-A2(+) cancer patients.
SO British journal of cancer, ***(2004 Apr 19)*** Vol. 90, No. 8, pp. 1563-71.
Journal code: 0370635. ISSN: 0007-0920.

L9 ANSWER 3 OF 10 MEDLINE on STN
TI Cytotoxicity of cord blood derived Her2/neu-specific ***cytotoxic*** ***T*** ***lymphocytes*** against human breast cancer in vitro and in vivo.
AB The Her2/neu oncogene encodes a transmembrane protein with homology to the ***epidermal*** ***growth*** ***factor*** ***receptor*** . Overexpression of this gene contributes to the aggressiveness of breast cancer and poor prognosis. Therefore, Her2/neu is an ideal target molecule for generating effective ***cytotoxic*** ***T*** ***lymphocytes*** (CTLs) against breast cancers. This study reports on the generation of Her2/neu-specific CTL from umbilical cord blood mononuclear cells (UCBC) using dendritic cells primed with Her2/neu-derived ***peptide*** (KIFGSLAFL, E75) for immunostimulation. The CTLs showed specific cytotoxicity to Her2/neu high expressing MDA-453 but not toward Her2/neu low expressing MDA-231 human breast cancer cells. Similarly generated CTLs stimulated with irrelevant ***peptide*** pulsed dendritic cells did not show significant cytotoxicity towards breast cancer targets. The phenotypes of cells in culture showed high percentage of CD3+, CD4+ and CD8+T cells as determined by flow cytometry. However, the ***antibody*** mediated blocking assay demonstrated that only HLA-Class I restricted CD8+ cells are involved in the cytotoxicity. Furthermore, in vivo studies showed that treatment of SCID mice bearing MDA-453 tumor with Her2/neu-specific CTLs resulted in significant inhibition of tumor growth compared to untreated tumor bearing control mice. These results demonstrate that human umbilical cord blood mononuclear cells are a good source for generating Her2/neu-specific CTLs against human breast cancer both in vitro and in vivo.
SO Breast cancer research and treatment, ***(2004 Jan)*** Vol. 83, No. 1, pp. 15-23.
Journal code: 8111104. ISSN: 0167-6806.

L9 ANSWER 4 OF 10 MEDLINE on STN
TI Cancer immunotherapy in head and neck region.
AB There is no one common immunotherapy for the treatment of head and neck cancer (H&N cancer). A streptococcal agent, OK-432, which is classified as a biological response modifier (BRM), is occasionally used by means of local administration for recurrent H&N cancer, and the response rate is approximately 18%. In regard to specific immunotherapy, a murine monoclonal ***antibody*** (named mAb 225) against the ***epidermal*** ***growth*** ***factor*** ***receptor*** (FGFR) that is frequently overexpressed in H&N cancer has been produced in the U.S.A. Furthermore, to obviate human anti-mouse ***antibody*** responses, a chimeric human-to-murine version of mAb 225 (C225) was developed by exchanging the constant regions of mAb 225 to counterparts in human immune globulin. Phase I clinical trials of C225 in the U.S.A. demonstrated that treatment with C225 was well tolerated and that C225 given in combination with cisplatin has biologic activity. On the other hand, many tumor antigens recognized by ***cytotoxic*** ***T*** ***lymphocytes*** (CTL) have been identified from a variety of malignant tumors and some of them, including the MAGE-3 antigen, are frequently expressed in H&N cancer. We identified an MAGE-3-derived epitope

recognized by HLA-A24-restricted CTL from peripheral blood mononuclear cells (PBMC). In contrast we failed to generate CTL specific for MAGE-3+/HLA-A24+ tumors from PBMC in any of 5 HLA-A24+ cancer patients whose tumors expressed the MAGE-3 gene. Therefore, we did not apply MAGE-3-derived CTL epitope in clinical uses such as ***peptide*** vaccine and ***peptide*** pulsed dendritic cell infusion for H&N cancer.

SO Gan to kagaku ryoho. Cancer & chemotherapy, ***(2001 Apr)*** Vol. 28, No. 4, pp. 461-6.
Journal code: 7810034. ISSN: 0385-0684.

L9 ANSWER 5 OF 10 MEDLINE on STN

TI HER-2/neu is expressed in human renal cell carcinoma at heterogeneous levels independently of tumor grading and staging and can be recognized by HLA-A2.1-restricted ***cytotoxic*** ***T*** ***lymphocytes*** .

AB The HER-2/neu oncoprotein, a 185 kDa membrane-associated tyrosine kinase with extensive homology to the ***epidermal*** ***growth*** ***factor*** ***receptor*** (EGF-R), is overexpressed in breast and ovarian carcinomas. Its overexpression is closely associated with poor prognosis in the course of disease. Here we demonstrate HER-2/neu overexpression in both established cell lines and biopsy material obtained from renal epithelial tumors. Immunohistochemical analysis of human kidney tumor lesions using 2 HER-2/neu-specific ***antibodies*** revealed HER-2/neu expression in more than 40% of primary epithelial renal tumors and more than 30% of primary renal cell carcinoma (RCC) specimens. A distinctive HER-2/neu expression pattern was found in different subtypes of kidney tumors with the highest frequency in chromophilic and chromophobic RCC, but neither associated with disease stage nor tumor grade. Eight of 10 RCC cell lines expressed significant levels of HER-2/neu mRNA and protein, but at a lower level compared with HER-2/neu overexpressing ovarian carcinoma cells. To evaluate the immune response against HER-2/neu expressing HLA-A2-positive (HLA-A2(+)) RCC cells, allogeneic HLA-A2-restricted ***cytotoxic*** ***T*** - ***lymphocyte*** (CTL) lines generated by pulsing dendritic cells with 3 different HER-2/neu-derived ***peptides*** , (HER-2(9.369), HER-2(9.435) and HER-2(9.689), were utilized in chromium-release assays. Specific lysis of HER-2/neu expressing HLA-A2(+) RCC cell lines was mediated by CTL lines specific for each of these 3 HER-2/neu-derived epitopes. The fine specificity of 2 CTL clones was defined to the epitopes HER-2(9.435) and HER-2(9.689). Their specificity was then confirmed by cold target inhibition assays. In addition, CTL-mediated lysis was enhanced by pulsing tumor cells with exogenous HER-2/neu-specific ***peptides*** . Our data suggest that (i) HER-2/neu is heterogeneously expressed in different subtypes of RCC, (ii) HER-2/neu is naturally processed by RCC and (iii) HER-2/neu epitopes presented by RCC can be recognized by HLA-A2-restricted, HER-2/neu-specific CTL.

SO International journal of cancer. Journal international du cancer, ***(2000 Aug 1)*** Vol. 87, No. 3, pp. 349-59.
Journal code: 0042124. ISSN: 0020-7136.

L9 ANSWER 6 OF 10 BIOSIS on STN

TI Immunobiology of HER-2/neu oncoprotein and its potential application in cancer immunotherapy.

AB HER-2/neu (also known as HER2 or c-erb-B2) is a 185-kDa protein receptor with tyrosine kinase activity and extensive homology to the epidermal growth factor (EGF) receptor. HER-2/neu is expressed in many epithelial tumors and known to be overexpressed in approximately 20-25% of all ovarian and breast cancers, 35-45% of all pancreatic adenocarcinomas, and up to 90% of colorectal carcinomas. HER-2/neu overexpression represents a marker of poor prognosis. HER-2/neu-positive tumor cells are potentially good targets for tumor-reactive ***cytotoxic*** ***T*** ***lymphocytes*** which have been utilized in immunotherapeutic trials. In addition, the "humanized" monoclonal ***antibody*** Herceptin has been tested in several clinical trials and proved to be an effective adjuvant therapy for HER-2/neu-positive breast and ovarian cancers. Vaccinations aiming at generating T-cell responses are being examined in both experimental and clinical trials. Natural immunity at the level of T and B cells has been observed in patients with HER-2/neu-positive tumors confirming the immunogenicity of HER-2/neu and encouraging vaccination trials with HER-2 protein-derived subunits or synthetic ***peptides*** . This review summarizes recent data from patients with various types of HER-2/neu-overexpressing cancers carrying different HLA alleles and exhibiting pre-existent immunity to HER-2/neu-derived synthetic ***peptides*** . It also discusses potential advantages of the various vaccination approaches to immunotherapy targeting the HER-2/neu molecule.

SO Cancer Immunology Immunotherapy, (***March 2004***) Vol. 53, No. 3, pp. 166-175. print.

CODEN: CIIMDN. ISSN: 0340-7004.

L9 ANSWER 7 OF 10 CAPLUS COPYRIGHT 2008 ACS on STN
TI Use of ***epidermal*** ***growth*** ***factor*** ***receptor*** -derived ***peptides*** recognized by both cellular and humoral immune responses in cancer patients
AB The present invention provides ***EGFR*** -derived ***peptides*** usable in ***EGFR*** -based immunotherapy for cancer. Namely, ***EGFR*** -derived ***peptides*** capable of inducing both cellular and humoral immune responses or a mutant ***peptide*** thereof; a polypeptide contg. the above ***peptide*** ; a nucleic acid mol. encoding the same; and a medicinal compn. such as cancer vaccines contg. the same; are provided. CTL recognizing HLA complex, and ***antibodies*** specific to those ***peptides*** , are also claimed. The ***epidermal*** ***growth*** ***factor*** ***receptor*** (***EGFR***) is one of the most appropriate target mols. for cancer therapy because of its high expression in epithelial cancers. ***Cytotoxic*** ***T*** ***lymphocyte*** (CTL)-directed epitope ***peptides*** could be a class of useful compds. in ***EGFR*** -targeted therapies. However, at present, there are no data on CTL-directed ***peptides*** of ***EGFR*** . Therefore, this study aimed to identify immunogenic ***EGFR*** -derived ***peptides*** in HLA-A24+ NSCLC patients. The authors report in this study three such ***EGFR*** -derived ***peptides*** at positions 54-62, 124-132 and 800-809. These ***peptides*** were recognized by both cellular and humoral immune responses in most of the peripheral blood mononuclear cells (PBMCs) and sera from NSCLC patients that we tested. The authors also report finding of two such types of ***EGFR*** -derived ***peptides*** at position 479-488 and 1138-1147, both of which were recognized by the majority of patients' sera (IgG), and also possessed the ability to induce HLA-A2-restricted ***peptide*** -specific CTLs against ***EGFR*** -pos. tumor cells in peripheral blood mononuclear cells (PBMCs) of epithelial cancer patients. These results may provide a scientific basis for the development of ***EGFR*** -based immunotherapy.
SO PCT Int. Appl., 39 pp.
CODEN: PIXXD2

L9 ANSWER 8 OF 10 CAPLUS COPYRIGHT 2008 ACS on STN
TI Non-affinity purification of proteins
AB A method is disclosed for purifying a target protein from a mixt. contg. a host cell protein, comprising subjecting said mixt. to: (a) a non-affinity purifn. step, followed by (b) high-performance tangential-flow filtration (HPTFF), and (c) isolating said protein in a purity contg. less than 100 ppm of said host cell protein, wherein said method includes no affinity purifn. step.
SO PCT Int. Appl., 77 pp.
CODEN: PIXXD2

L9 ANSWER 9 OF 10 CAPLUS COPYRIGHT 2008 ACS on STN
TI Novel methods for therapeutic vaccination
AB A method is disclosed for inducing cell-mediated immunity against cellular antigens. More specifically, the invention provides for a method for inducing ***cytotoxic*** ***T*** - ***lymphocyte*** immunity against weak antigens, notably self-proteins. The method entails that antigen presenting cells are induced to present at least one CTL epitope of the weak antigen and at the same time presenting at least one foreign T-helper lymphocyte epitope. In a preferred embodiment, the antigen is a cancer specific antigen, e.g. prostate specific membrane antigen (PSM), Her2, or FGF8b. The method can be exercised by using traditional polypeptide vaccination, but also by using live attenuated vaccines or nucleic acid vaccination. The invention furthermore provides immunogenic analogs of PSM, Her2 and FGF8b, as well as nucleic acid mols. encoding these analogs. Also vectors and transformed cells are disclosed. The invention also provides for a method for identification of immunogenic analogs of weak or non-immunogenic antigens.
SO PCT Int. Appl., 220 pp.
CODEN: PIXXD2

L9 ANSWER 10 OF 10 CAPLUS COPYRIGHT 2008 ACS on STN
TI Synergistic composition and methods for treating neoplastic or cancerous growths and for restoring or boosting hematopoiesis
AB A method for treating neoplastic or cancerous growths and for treating cancer patients to restore or boost hematopoiesis comprises administration of a combination of a ***cytotoxic*** ***T*** - ***lymphocyte*** (CTL)-inducing compn. and .gtoreq.1 agent capable of neutralizing or down-regulating the activity of tumor-secreted immunosuppressive factors such as TGF-.beta. and IL-10, sep. or in combination. The CTL inducer is typically a vaccine for enhancing tumor immunity which lacks an

immunostimulating ***peptide*** component and is formulated as a stable oil-in-water emulsion contg. a micelle-forming agent. The combination produces a synergistic enhancement of the CTL response. Since TGF-.beta. neg. regulates and/or inhibits the growth of hematopoietic cells, the treatment can improve hematopoiesis during cancer therapy. Thus, mice bearing progressively growing ovalbumin-expressing EG7 tumors showed a delay in tumor growth after treatment with 30 .mu.g ovalbumin in Provax adjuvant and 50 .mu.g anti-TGF-.beta. ***antibodies*** .

SO PCT Int. Appl., 41 pp.
CODEN: PIXXD2

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L13          9 L9 AND 1970-2004/PY

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L2        53835 "EPIDERMAL GROWTH FACTOR RECEPTOR"
L3        62793 L1 OR L2
L4        48904 "CYTOTOXIC T LYMPHOCYTE"
L5           79 L3 AND L4
L6           55 L5 AND 1970-2005/PY
L7           29 L6 AND ANTIBOD?
L8           18 DUP REM L7 (11 DUPLICATES REMOVED)
L9           10 L8 AND PEPTIDE
L10          19 KYOGO?/AU AND ITOH?/AU
L11           0 SHIGEKI?/AU AND SHICHIJO?/AU
L12           0 L9 AND L10
L13           9 L9 AND 1970-2004/PY

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